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EFFECTS OF COUGHING ON INTRATHORACIC PRESSURE, ARTERIAL PRESSURE AND PERIPHERAL BLOOD FLOW

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Pulse pressure, as well as mean pressure, changes have been shown to influence baroceptors (Ead, Green & Neil, 1952). In man, rapid pressure fluctuations or transients in the vascular system are derived from the heart beat (pulse pressure) or from external factors. One of the latter is coughing, and 'cough transients' of large amplitude can be imposed on the arterial, cardiac and venous pressure pulses. Some of their effects on the circulation and lungs are described in this paper.

Since both coughing and the Valsalva manoeuvre raise intrathoracic pressure it has been found convenient to compare the two procedures.

METHODS

Fifty-three normal subjects were investigated. Intrathoracic pressure was measured with an intraoesophageal water-filled polythene tube attached to a capacitance manometer (Dornhorst & Leathart, 1952) and intravascular pressures were also measured with capacitance manometers (Hansen, 1949). The Valsalva manoeuvre was performed by blowing a mercury column to 40–60 mm Hg and holding it at that level for about 10 sec. Forearm flow was measured with a water-filled (34° C) venous-occlusion plethysmograph and recorded simultaneously with the pressure curves.

RESULTS

Intrathoracic pressure during coughing. Ordinary intermittent coughing consists of an explosive expiratory effort followed by a rapid inspiration and further expiratory effort. Intrathoracic pressure records showed transients of 100–250 mm Hg according to the violence of the cough (Fig. 1). The duration of such transients was usually about one second.

The intrathoracic pressure changes of coughing and the Valsalva manoeuvre were transmitted to the peripheral arteries, veins without valves, and chambers of the heart (Fig. 1). The pulse pressure decreased during coughing, as well as during the Valsalva manoeuvre. This decrease in pulse pressure is not visible in the record shown in Fig. 2, since it is obscured by the large cough transients; in many other records, however, it could be observed.

Arterial blood pressure and forearm flow following coughing. On ceasing intermittent coughing, arterial pressure fell below the control level, pulse pressure was reduced and the heart rate increased in all subjects. Blood pressure then rose gradually to the control level (Fig. 2). During the early part of this period forearm flow was increased, indicating vasodilatation of muscle vessels (Fig. 3).

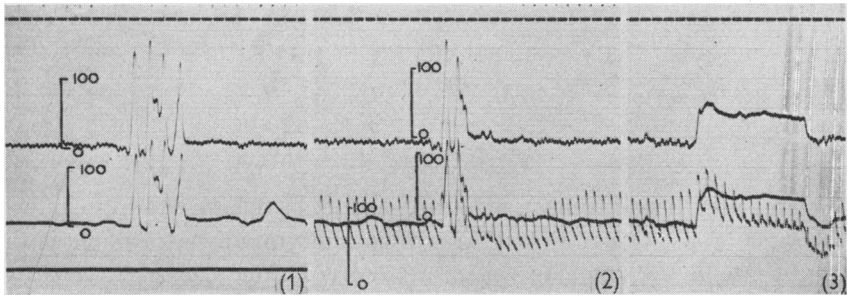

Fig. 1. Upper curve, right auricular pressure. Lower curve intra-oesophageal pressure with arterial pressure superimposed in records (2) and (3). Four cough transients are shown in the first record, two in the second and the effect of the Valsalva manoeuvre in the third. Calibration in mm Hg and time marker in seconds in this and subsequent records.

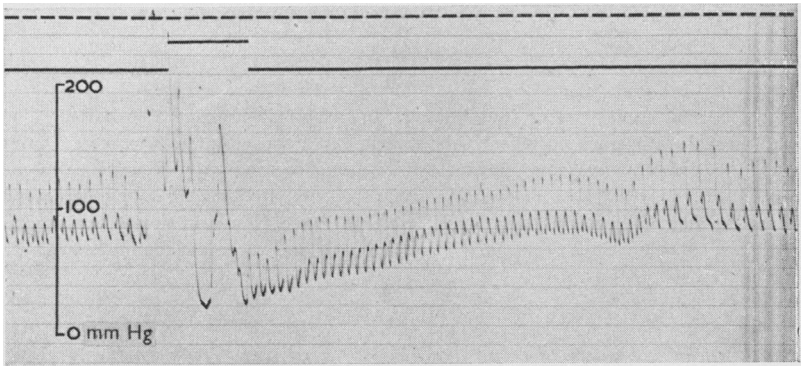

Fig. 2. Arterial pressure showing effect of a few large cough transients.

In contrast, following the Valsalva manoeuvre, there was a rise of blood pressure, bradycardia, and constriction in the forearm (Fig. 4).

Blood flow changes in a sympathectomized limb. Two subjects with sympathectomized upper limbs were studied. The forearm flow followed arterial blood pressure, increasing during the overshoot after the Valsalva manoeuvre and decreasing during hypotension following coughing. Vasodilatation following coughing in normal limbs was therefore mediated through the vasomotor nerves.

Intrathoracic and cardiac pressures following cessation of coughing. In early experiments it was found that right ventricular diastolic and right auricular pressures remained elevated 6–10 mm Hg above resting level for 5–70 sec following cessation of intermittent coughing. It seemed possible that constriction in the pulmonary arterial system might be responsible since pul-

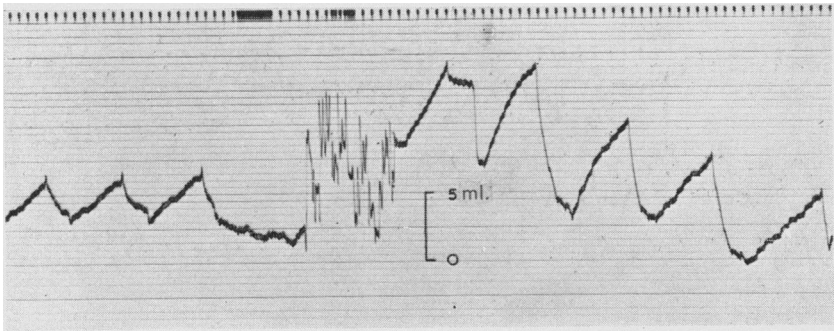

Fig. 3. Forearm blood flow, showing increase in flow after coughing, indicated by artifacts on the trace. The rate of volume increase (proportional to forearm flow) on inflating a collecting cuff, is recorded three times before and five times after coughing.

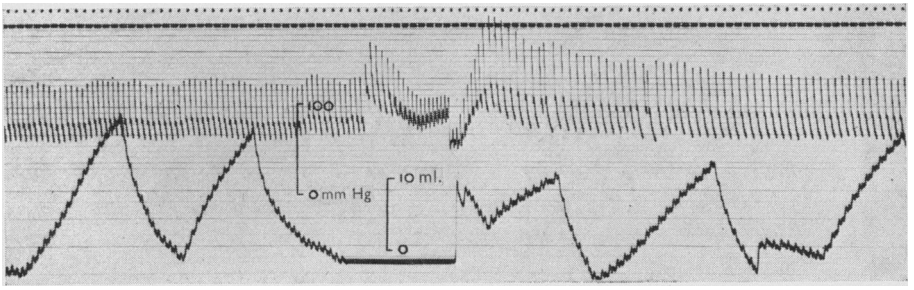

Fig. 4. Upper curve arterial pressure. Lower curve forearm blood flow. Showing the effect of the Valsalva manoeuvre with constriction in the forearm during the arterial 'overshoot'. The onset of the Valsalva manoeuvre is shown on the arterial pressure trace by a sudden increase of about 40 mm Hg and the end by a sudden decrease 11 sec later.

monary arterial diastolic and, to a lesser extent, systolic pressures were also raised. When intrathoracic pressure was measured simultaneously, however, it was found to be elevated to the same extent and to fall to the resting level in the same manner (Fig. 5). The residual elevation of intracardiac pressures therefore resulted from the rise of intrathoracic pressure. This post-tussive elevation of intrathoracic pressure was observed in all subjects who coughed intermittently and was absent following the Valsalva manoeuvre (Fig. 6). The

latter procedure was followed by an immediate return of intrathoracic and cardiac pressures to control levels. The difference in behaviour of intrathoracic pressure after coughing and after the Valsalva manoeuvre is also shown in Figs. 1 and 7 on pressure traces of smaller amplification.

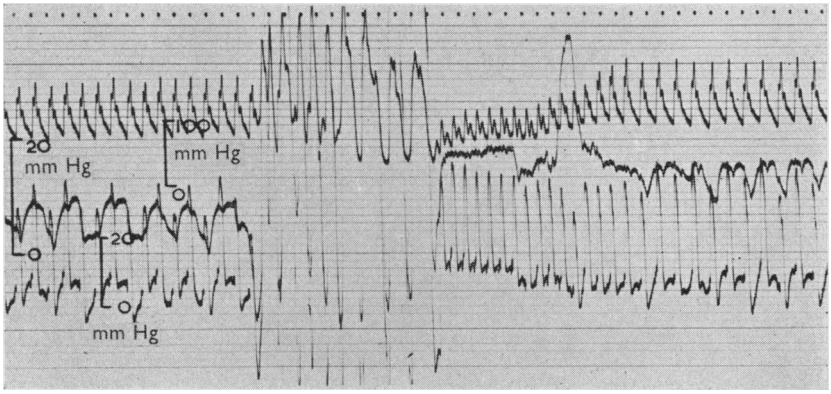

Fig. 5. Upper curve, arterial pressure. Middle curve intra-oesophageal pressure. Lower curve, right ventricular pressure. Following coughing, arterial pressure is reduced and oesophageal and right ventricular diastolic pressures increased.

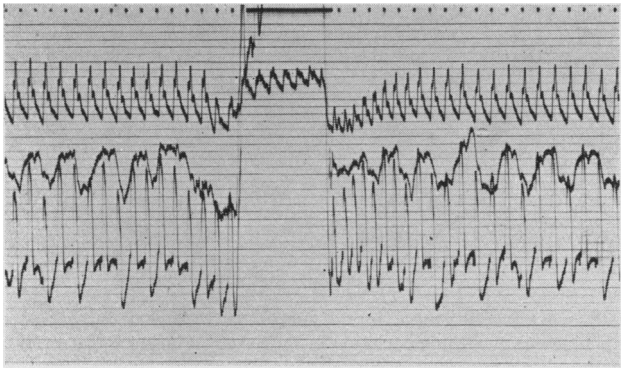

Fig. 6. From the same continuous record as Fig. 5. The Valsalva manoeuvre is shown by the gap in the oesophageal and right ventricular pressure traces which rise beyond the top of the recording paper. After the Valsalva manoeuvre intra-oesophageal and right ventricular diastolic pressures return immediately to control level.

Degree of response. The circulatory and intrathoracic pressure changes after coughing were proportional to the violence of the cough. While this was true of the fifty-three subjects as a whole, it was best studied in the individual subject by repeated observations over a short period (Fig. 7). The greatest

intrathoracic pressure changes (over 300 mm Hg) were achieved by certain middle-aged males during spontaneous coughing. The subsequent hypotension was sometimes sufficient to cause syncope (Sharpey-Schafer, 1953).

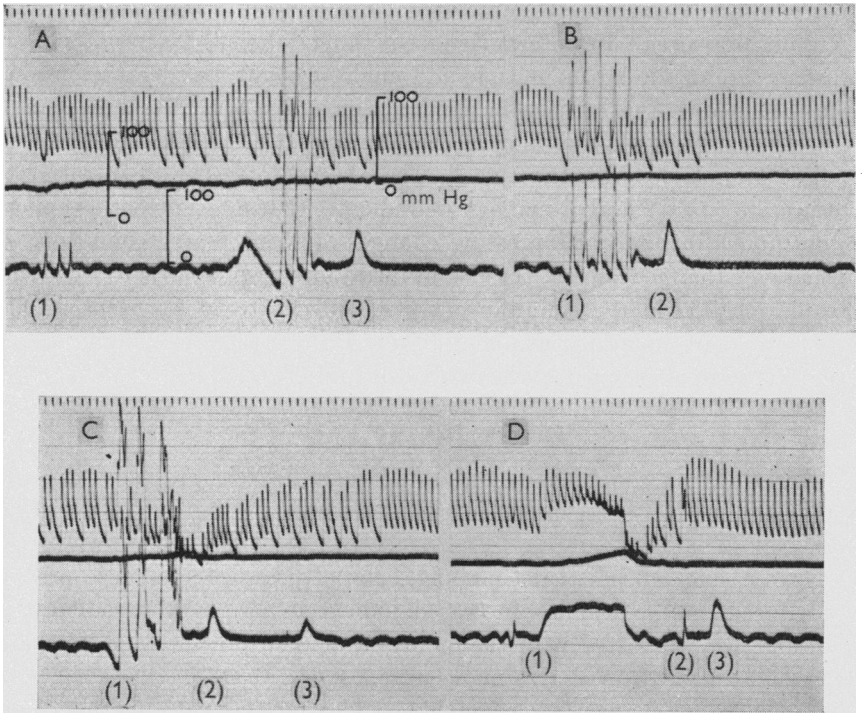

Fig. 7. Parts of a continuous record. A middle-aged subject showing frequent ectopic beats. Upper curve, arterial pressure. Middle curve, pressure in a forearm vein. Lower curve, intra-oesophageal pressure. *A*: (1) three 'ahems'; (2) three coughs; (3) swallow. *B*: (1) five coughs; (2) swallow. *C*: (1) three stronger coughs; (2) and (3) swallows. *D*: (1) Valsalva manoeuvre; (2) small cough; (3) swallow.

DISCUSSION

The different circulatory effects of coughing and the Valsalva manoeuvre can be explained as reflex changes from stimulation of receptors by vascular pressure transients. Thus vasodilatation follows the large cough transients while vasoconstriction follows the diminished pulse pressure occurring during the Valsalva manoeuvre. Ead *et al.* (1952), using the isolated perfused carotid sinus of the dog, showed that systemic arterial pressure fell when perfusion pulse pressure was increased and rose when perfusion became non-pulsatile, the mean perfusion pressure being kept constant throughout. In man it has not yet proved possible to locate the receptors stimulated by coughing.

The rise in intrathoracic pressure after intermittent coughing is unlikely to be due to accumulation of blood in the chest or to muscular straining as normal respiration can be observed during this period. More probably, air is trapped temporarily in the lungs. Daly & Schweitzer (1951) demonstrated bronchoconstriction in dogs when the isolated carotid sinus was exposed to high perfusion pressures. In intermittent coughing, observed arterial pressure transients are of similar magnitude and might account for reflex retention of air in the alveoli. Another possible explanation is that the effect is mechanical, air being trapped by a valvular mechanism operating at the opening of the smallest bronchioles into the alveoli. The absence of a rise of intrathoracic pressure after the Valsalva manoeuvre indicates that intermittent as opposed to continuous intrathoracic pressure changes are a necessary condition.

Whatever the mechanism, the rise in intrathoracic pressure after coughing may play a part in the simultaneously recorded arterial hypotension since positive pressure breathing at about 10 mm Hg decreases the filling pressure of the heart and consequently the cardiac output (Werkö, 1947).

SUMMARY

1. Intermittent coughing caused rapid intrathoracic pressure transients up to 250 mm Hg. These pressure changes were transmitted to peripheral arteries.

2. On ceasing intermittent coughing, arterial pressure and pulse pressure were reduced and forearm flow increased, indicating peripheral vasodilatation. These findings contrasted with the increased arterial pressure and decreased forearm flow after the Valsalva manoeuvre.

The blood flow changes after coughing and after the Valsalva manoeuvre did not occur in sympathectomized limbs.

The results are interpreted in terms of the effect of large (coughing) and small (Valsalva) arterial pressure transients on baroceptors.

3. Intrathoracic pressure remained elevated (6–10 mm Hg) for many seconds after intermittent coughing. This did not occur after the Valsalva manoeuvre. It is suggested that air may be trapped in the lungs, possibly by reflex bronchoconstriction. The residual raised intrathoracic pressure may play a part in the hypotension which follows coughing.

REFERENCES

DALY, M. DE BURGH & SCHWEITZER, A. (1951). Reflex bronchomotor responses to stimulation of receptors in the regions of the carotid sinus and arch of the aorta in the dog and cat. *J. Physiol.* **113**, 442–462.

DORNHORST, A. C. & LEATHART, G. L. (1952). A method of assessing the mechanical properties of lungs and air-passages. *Lancet*, ii, 109.

EAD, H. W., GREEN, J. H. & NEIL, E. (1952). A comparison of the effects of pulsatile and non-pulsatile blood flow through the carotid sinus on the reflexogenic activity of the sinus baroceptors in the cat. *J. Physiol.* **118**, 509–519.

HANSEN, A. T. (1949). Pressure measurement in the human organism. *Acta. physiol. scand.* **19**, Suppl. 68.

SHARPEY-SCHAFER, E. P. (1953). The mechanism of syncope after coughing. *Brit. Med. J.* ii, p. 860.

WERKÖ, L. (1947). The influence of positive pressure breathing on the circulation in man. *Acta. med. scand.* **128**, Suppl. 193.